Coccidiosis in guinea-pigs

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SYNOPSIS The attention of laboratory workers is drawn to the possibility of coccidiosis as a cause of death in guinea-pigs. The purchase of a number of guinea-pigs infected with this protozoon was followed by 12 deaths when these animals were injected with material for diagnostic purposes. No deaths occurred in the laboratory stock herd, as these were kept separate from the newcomers and were not infected.

The life history of the parasite is described, together with the post-mortem findings in our series of animals.

MATERIAL AND METHODS

Because of an increase in the number of requests for biological examinations in April 1960, a stock of guinea-pigs was purchased and used without a preliminary quarantine period. These animals received an intramuscular injection into the right thigh of material such as uterine curettings, urine, or milk, and of those injected 12 subsequently died. All the deaths occurred among the imported animals and followed a brief illness in which diarrhoea was the principal symptom. The imported guinea-pigs were kept separate from the laboratory stock and no deaths or illness occurred in the latter group.

POST-MORTEM EXAMINATION

Post-mortem examination, together with bacteriological investigations, were performed on all guinea-pigs dying and, in addition, material was frozen and kept in case virological examination might be required.

Direct wet preparations of faeces were examined for parasites and cysts were sought for after concentration by the salt flotation method.

Material for histological examination was fixed in formalin and sections were stained by haematoxylin and eosin. A comprehensive histological examination was performed on only three of the guinea-pigs.

RESULTS

GROSS APPEARANCE OF ORGANS The visceral peritoneum was congested and there was bloodstained fluid in the peritoneal cavity. White patches on the outer surface of the colon were taken to be patches of lymphoid tissue.

BACTERIOLOGY Bacteriological examination was

negative and in view of the findings described below virological studies were not undertaken.

HISTOLOGY The only abnormal findings outside the intestinal tract were areas of pulmonary collapse.

The visceral peritoneum and mesentery in relation to the gut showed a non-specific inflammatory infiltration consisting of a mixture of polymorphs and chronic inflammatory cells.

The whitish patches on the colon were areas of lymphoid tissue, some of which showed ulceration with a surrounding inflammatory reaction. Ulceration appeared particularly liable to occur within small mucosal diverticuli and, in sections not passing through the opening of the diverticulum, produced the appearance of abscess cavities lying within the submucosa. The mucosa of the colon near these lesions contained enormous numbers of cysts measuring $18 \times 15 \mu$ which were identified as the oocysts of the protozoal parasite *Eimeria caviae*. The cysts had a well-defined capsule and contained a mass of granular cytoplasm.

In one case no ulceration was found and only a few degenerated oocysts were present in the colon, together with a non-specific enteritis affecting most of the small intestine.

In the infected animals, three further stages in the life cycle of *Eimeria caviae* were found in the deeper parts of the mucosa: (1) the schizont, (2) the macrogametocyte, and (3) the microgametocyte.

THE LIFE CYCLE OF THE ORGANISM

Maturation of oocysts was studied by emulsifying a small portion of faeces in 5% sodium dichromate

FIG. 1

FIG. 2

FIG. 1. Three oocysts, one of which is in the process of division into four sporocysts.

FIG. 2. A section of large intestine containing three parasites. The centre one, with a prominent nucleolus, is a macrogametocyte (♀). Near to this is a microgametocyte (♂) showing nuclear division into microgametes.

and placing it in a Petri dish at room temperature for a period of four to five days. At the end of this time the oocysts showed division into four distinct masses (Fig. 1) representing the four sporocysts characteristic of the genus *Eimeria*. In the natural cycle this mature oocyst is ingested and the sporocysts each release a pair of sporozoites which escape to infect the epithelial cells of the large intestine.

The first, asexual, stage of reproduction, that of schizogony, then takes place and is followed by the liberation of merozoites capable of entering more epithelial cells and repeating the asexual cycle.

The asexual forms appear as inclusions within the cytoplasm of epithelial cells, young individuals being smaller than the cell nucleus. Older parasites are larger and show several nuclei. At a later stage a sexual process occurs, microgametocytes (male) and macrogametocytes (female) being produced (Fig. 2).

The macrogametocyte is a prominent cell with a round nucleus containing a small chromatin condensation. Within the cytoplasm are numerous coarse granules.

The microgametocyte is of similar size but instead of a single discrete nucleus it contains a large number of elongated or comma-shaped nuclear bodies which represent the microgametes. Rupture of the microgametocyte releases the microgametes which, by means of their flagellae, swim to the macrogametocytes which they fertilize. After fertilization the cytoplasmic granules of the macrogametocytes fuse to produce the oocyst capsule. The oocyst is passed in the faeces and matures outside the body of the host. On being ingested by a suitable host the sporozoites are liberated and the asexual cycle is repeated.

DISCUSSION

Coccidiosis is due to infection of the alimentary tract with protozoa of the genus *Eimeria*, class Sporozoa, all members of which are obligatory intracellular parasites. Within the genus there is a large number of species all showing a high degree of host specificity. Practically all domestic animals are potential hosts for *Eimeriae* but coccidiosis is best

known as a disease of poultry and rabbits in which it may reach epidemic proportions resulting in serious economic loss. Man is not a host for any species of *Eimeria* although the closely related *Isospora* occasionally causes a dysentery-like illness. Those engaged in medical laboratory work are therefore apt to be unfamiliar with the disease.

Infection of guinea-pigs with *Eimeria caviae*, whose full life cycle was first described by Sheather (1924), was at one time considered to be extremely common but of little consequence. This view, endorsed by Henry (1932) and Lapage (1940), is also expressed in textbooks of protozoology such as that of Wenyon (1926). It is possible that the care taken in the maintenance of laboratory guinea-pigs in recent years has resulted in a decrease in the incidence of infection. Under experimental conditions, however, healthy guinea-pigs can be infected by feeding with washed suspensions of cysts (Henry, 1932). If the dose is large, clinical illness may follow with diarrhoea and eventually death. In the natural course of events death does not usually ensue and it would appear that it can do so only when infection is heavy and is combined with other factors which upset the balance between the organism and its host.

The recent importation into this laboratory of a

number of infected guinea-pigs appears to have provided the necessary upset in equilibrium. In this instance, transportation, the introduction to new surroundings, different food, and finally the intramuscular injection of clinical material for diagnostic purposes, may have had some bearing on the appearance of intestinal ulceration with secondary infection, diarrhoea, and eventual death.

Once the diagnosis was established we were able to confirm that the imported guinea-pigs were infected and although these had been kept separate from our breeding herds we treated all guinea-pigs by adding sulphamezathine to their drinking water. An interrupted course of treatment was used in the form of three days' treatment and two days' rest, repeated to a total of nine days' treatment, and proved successful.

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REFERENCES

- Henry, D. P. (1932). *Univ. Calif. Publ. Zool.*, 37, 211.
 Lapage, G. (1940). *Vet. J.*, 96, 144, 190, 242, 280.
 Sheather, A. L. (1924). *J. comp. Path.*, 37, 243.
 Wenyon, C. M. (1926). *Protozoology*, Vol. 2. Baillière, Tindall & Cox, London.